improper form. This response is timely filed, as a Petition for a Three Month Extension of Time, with a check for the required fee of $460.00, is submitted herewith, making the response due on October 24, 2001. Applicants respectfully request reconsideration and withdrawal of the rejections in view of the following amendments and remarks.

A version of the amended paragraphs of the specification is attached hereto as Appendix A, and a version of the amended claims is attached hereto as Appendix B, with the changes made by the present amendment indicated in bold.

AMENDMENTS

Amendments to the Specification

Please amend the first full paragraph on page 15, at lines 3-28, of the specification so as to read as follows:

A1 SUB B3

Figs. 5 (A) and 5 (B) The amino acid sequences of human BPI (SEQ ID NO: 3), LBP (SEQ ID NO: 4), PLTP (SEQ ID NO: 5), and CETP (SEQ ID NO: 6). The alignment was performed with CLUSTAL [D. G. Higgins and P. M. Sharp, Gene, 73:237 (1989)] using all eleven known protein sequences from mammals [R. R. Schuman, et al., Science, 249:1429 (1990); D. Drayna et al., Nature, 327:632 (1987); R. Day et al., J. Biol. Chem., 269:9388 (1994); S. R. Leong and T. Camerato, Nucleic Acids Res., 18:3052 (1990); M. Nagashima, J. W. McLean, R. M. Lawn, J. Lipid Res., 29:1643 (1988); M. E. Pape, E. F. Rehber, K. R. Marotti, G. W. Melchior, Artheriosclerosis 11:1759 (1991); G. Su et al., J. Immunol., 153:743 (1994); P. W. Gray et al., J. Biol. Chem. 264: 9505 (1989); Albers et al., Biochem. Biophys. Acta, 1258:27 (1995); X. C. Jiang et al., Biochemistry, 34:7258 (1995); L. B. Agellon et al., Biochemistry, 29:1372 (1990); X. C. Jiang et al., J. Biol. Chem., 266:4631 (1991)] but only the four human sequences are shown. Residues that are completely conserved in all proteins are indicated below the sequence *; those which are highly conserved are indicated by •. The secondary structure of BPI is indicated above the sequences. The strands are indicated by arrows; strands which make up the central β sheet are shown

with gray arrows. Because of the β bulges and pronounced twisting, some of the β strands have one or more residues that do not show classical H-bonding patterns or ΦΨ angles; these breaks are indicated by ^ above the strands. The α helices are shown as cylinders, and one-residue breaks in helices B and B' are indicated with a vertical dashed line. The horizontal dashed line indicates the linker region. Peptides from BPI and LBP with the highest lipopolysaccharide-binding activity (Little, et al., J. Biol. Chem. 268: 1865 (1994); Taylor et al., J. Biol. Chem. 270: 17934 (1995)) are in bold italics. The disulfide bond is indicated by S-S. Residues with atoms within 4 Å of the H_2 -terminal lipid are highlighted with gray shading; residues within 4 Å of the COOH-terminal lipid are shown with white letters in black boxes.

Please amend the first full paragraph on page 44 of the specification so as to read as follows:

The glycosylation site was next removed by replacing the region from a unique XcmI site to a unique SphI site within the BPI gene in pSS101 with an annealed oligonucleotide that contained the codon (TCC) for the serine at amino acid position 351 changed to the codon (GCC) for alanine as shown below.

```
Wild type
     XcmI                                              SphI
...CCC AAC TCC TCC CTG GCT TCC CTC TTC CTG ATT GGC ATG CAC (SEQ ID NO:7)
...GGG TTG AGG AGG GAC CGA AGG GAG AAG GAC TAA CCG TAC GTG (SEQ ID NO:8)
   Pro Asn Ser Ser Leu Ala Ser Leu Phe Leu Ile Gly Met His (SEQ ID NO:9)
                 351
Nonglycosylated
     XcmI                                              SphI
...CCC AAC TCC GCC CTG GCT TCC CTC TTC CTG ATT GGC ATG CAC (SEQ ID NO:10)
...GGG TTG AGG CGG GAC CGA AGG GAG AAG GAC TAA CCG TAC GTG (SEQ ID NO:11)
   Pro Asn Ser Ala Leu Ala Ser Leu Phe Leu Ile Gly Met His (SEQ ID NO:12)
                 351
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This step generated the plasmid pSS102.